

EXHIBIT 6

Expert Opinion



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This report details my medical and scientific opinion regarding whether there is a relationship between Vioxx and ischemic cardiovascular disease. I have also been asked to comment on the cardiovascular history of a patient, Jo Levitt. Details of my education, professional experience and list of publications are in my Curriculum Vitae.

1. I have been asked to provide my medical and scientific opinion as to whether there is a relationship between non-steroidal anti-inflammatory drugs (NSAIDs), including Vioxx, and ischemic cardiovascular disease. Below is my review of medical and scientific issues that are relevant to a consideration of this question. I have reviewed the literature on the subject, paying particular attention to evidence dealing with the biological plausibility of this potential relationship and the level of certainty with which one might conclude that such a relationship exists based on the evidence gathered from controlled clinical trials. I have also been asked to comment on the medical records for a specific patient, detailed below. Finally, I have been asked to address the state of the science while Vioxx was on the market.

Section I - Background and Qualifications

2. I graduated from the University of Oklahoma in 1976 with a BA in Psychology and earned my Medical Degree from the University of Texas Southwestern Medical School in 1980. I trained in Internal Medicine at Parkland Memorial Hospital in Dallas, where I was a Chief Medical Resident. I did my fellowship training in Cardiology at Brigham and Women's Hospital, Harvard Medical School, and during this time I began my research in the area of thrombosis. I completed additional post-doctoral training at the Center for Thrombosis and Vascular Research, University of Leuven, Belgium.
3. After completing my training, I was appointed as an Instructor at Harvard Medical School and was promoted to Assistant Professor of Medicine in 1990. Until 1993, I served as Co-Director for the Center for Research in Thrombolysis at Brigham and Women's Hospital in Boston. I was recruited to Vanderbilt Medical School and appointed as an Associate Professor of Medicine and Pharmacology at Vanderbilt

the evidence, the data linking Vioxx with increased cardiovascular risk fail to meet currently accepted, evidence-based standards of practice. Furthermore, the totality of the data fails to establish causality.

Section VII – Jo Levitt

71. I have been provided medical records pertaining to Jo Levitt and in this section of my report I will review her cardiovascular history and comment on specific details of note in this case. Ms. Levitt is a 72 year-old woman (DOB: 30 June 1943). On 9 March 2000, at the age of 56, Ms. Levitt was hospitalized for an acute coronary syndrome. Coronary arteriography revealed a high grade narrowing of the left anterior descending coronary artery that involved her first diagonal branch while her other two coronary arteries were reportedly free of obstructive lesions. She had a stent placed in her left anterior descending artery and the ostium of the first diagonal branch was treated with balloon angioplasty without stenting (although the medical records are not completely uniform in this regard). Thereafter, in May 2000, Ms. Levitt again complained of chest pain. On May 26, 2000, Ms. Levitt was found to have in-stent restenosis and was treated with bypass surgery on May 28, 2000. Ms. Levitt never suffered a myocardial infarction, and serial cardiac exams of different types have confirmed a normal functioning heart and normal exercise capacity on numerous occasions.
72. Prior to her acute coronary syndrome episode in March 2000, Ms. Levitt suffered from depression, anxiety, function-limiting musculoskeletal pain and degenerative disk disease. She was repetitively noted to have chronic fatigue and sleep disorder, and in October 1999 was also diagnosed with fibromyalgia. Her medical records suggest that she had tried multiple NSAIDs over the decade prior, including, ibuprofen, peroxicam, naproxen, and aspirin to attempt to relieve her chronic pain.
73. In the week before her admission on 9 March 2000, Ms. Levitt complained of progressive angina-like symptoms that occurred upon mild exertion and were relieved by rest. Approximately one week before her admission, she was advised by the emergency medical team to go to the hospital, but she elected not to follow their advice. On the morning of her admission, she was awakened by chest discomfort. She presented to Dr. Hartman's office (her primary care physician) and, after an EKG was performed, she was immediately transferred by ambulance to the emergency room. Notably, immediately prior to her transfer, Ms. Levitt was given nitroglycerin and "within a minute had facial flushing and relief of her chest pressure and

81.7 Thus, based on Ms. Levitt's risk profile, she was at elevated risk compared with an otherwise healthy 56 year old woman for suffering a cardiovascular event.

82. As referenced above, in March 2000, Ms. Levitt presented with an episode of unstable angina, not a myocardial infarction. Moreover, prior to her presentation to the hospital, Ms. Levitt was provided nitroglycerin and had relief of her chest pressure and discomfort. The fact that her symptoms were relieved with nitroglycerin suggests that she had some component of coronary vasospasm or an acute manifestation of endothelial dysfunction that triggered her episode on 9 March 2010. As detailed above (section IVa), Vioxx does not have any adverse effect on vascular tone or endothelial function.

83. Further, the data with respect to any association between Vioxx and the specific syndrome suffered by Ms. Levitt – unstable angina / acute coronary syndrome – are weak. With respect to the major trials and data points, the available evidence does not suggest that Vioxx is associated with an increased risk of UA (numbers are total UA events and events per 1000 patient years):

83.1 VIGOR (50mg v. naproxen): Vioxx (5) (1.85) v. Naproxen (3) (1.11). This trial used twice the dose of Vioxx than used by Ms. Levitt.

83.2 ADVANTAGE (25mg v. naproxen): Vioxx (1) (1.56) v. Naproxen (2) (3.18).

83.3 March 2004 Pooled Analyses of Vioxx Clinical Trials: Placebo Comparison: Vioxx (10) (3.31) v. Placebo (10) (3.57); Non-Naproxen NSAIDs Comparison: Vioxx (9) (3.24) v. Non-naproxen NSAIDs (6) (4.19); Naproxen Comparison: Vioxx (10) (1.68) v. Naproxen (8) (1.84) [naproxen comparison includes VIGOR and ADVANTAGE above].

83.4 APPROVe: On-Drug + 14 days: Vioxx (7) (2.29) v. Placebo (4) (1.20), p=0.46 (NS); ITT censored on last day at risk: Vioxx (8) (1.5) v. Placebo (9) (1.7).

83.5 ViP: Vioxx (4) (3.64) v. Placebo (2) (1.81); and VICTOR: Vioxx (1) (1.08) v. Placebo (1) (1.01).

83.6 None of the comparisons, whether viewed individually or collectively or against placebo or active comparators, suggest an increased association between Vioxx and the specific syndrome of unstable angina. In particular, the pooled analyses of active comparators do not provide evidence of an increased risk attributable to Vioxx (again, based on her history, Ms. Levitt's treatment choice was likely another NSAID, not lack of treatment).

84. In addition to the fact that the symptoms of unstable angina on 9 March 2010 were relieved by taking nitroglycerin (again, suggesting some component of vasospasm), in